BOOK REVIEWS

THE WESTERN JOURNAL OF MEDICINE does not review all books sent to it by the publishers. A list of new books received is carried in the Advertising Section.

HAZY...? CRAZY...? AND/OR LAZY...?—The Maligning of Children with Learning Disabilities—Joseph H. Rosenthal, MD, Director, Learning Disabilities Clinic, Department of Pediatrics, Kaiser-Permanente Medical Clinic, Oakland. Academic Therapy Publications, 1539 Fourth Street, San Rafael (94901), 1973. 210 pages, $3.75.

A good physician must learn what he knows and what he doesn't know, what he is and what he is not. Having determined that he is a pediatrician, not a psychiatrist or a school teacher or counselor or a psychologist or a psychometrist, the physician should find Dr. Rosenthal's book of great value and support. It presents an individualized approach that takes advantage of an encyclopedic familiarity with the literature.

There is a good review of the assorted categories and diagnoses used, recognizing their limited meaning. Dr. Rosenthal recognizes that children come with parents and with schools, and none are "all o.k." or "all not o.k." He also accepts the expectations that middle-class America has of its schools and its children . . . a lot!

Perhaps one of the most useful concepts is the thesis that there are children who are inherently "different" (organic), and that many school, social and family problems are secondary and/or interrelated. Many mothers need help and rest, not more guilt or psychiatric counseling. The book is practical, offering management help by examples, as well as through more comprehensive diagnosis.

It is a good book, contains an immense amount of information in a small package, and is the intensely personal experience of a particular kind of well-trained and patient care oriented pediatrician. In a new field, where none of us is always sure of his ground, the well-trained pediatrician who wants to help more of the children in his practice will find Dr. Rosenthal's experiences interesting, useful and entertaining.

HENRY RICHANBACH, MD

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THE PHYSIOLOGICAL AND CELLULAR BASIS OF METABOLIC BONE DISEASE—Howard Rasmussen, MD, PhD, Benjamin Rush Professor of Biochemistry, University of Pennsylvania School of Medicine and Senior Physician, Children's Hospital of Philadelphia; and Philippe Bordier, MD, PhD, Maitre de Recherche-INSERM, Unité de Recherche "André LICHTWITZ" Hopital Lariboisière and Rhumatologue Attaché à la Clinique Rhumatologique de l'Hopital Lariboisière (Pr. De Seze), Université Paris Nord, Paris. The Williams & Wilkins Company, 428 E. Preston St., Baltimore (21202), 1974. 364 pages, $21.50.

This clearly written, beautifully illustrated and well-edited volume presents an attractive and interesting new approach to the understanding of skeletal physiology and pathophysiology. The authors' views are derived from imaginative interpretation of specially prepared bone biopsy specimens, mostly human and some from well-chosen animal experiments. They also review pertinent biochemistry, including recent information about vitamin D metabolism but not the heterogeneity of the parathyroid hormones.

The cellular basis of bone physiology used to be simple: osteoblasts built bone and osteoclasts destroyed it. Each was assumed to arise from a separate cell line. Then in 1963 Bélanger established the rôle of osteocytes in osteolysis, the principal process responsible for maintenance of blood calcium levels.

In this work, Rasmussen and Bordier present new evidence that osteoprogenitor cells (mesenchymal cells, osteocytes, resting osteoblasts) differentiate to become osteoclasts which in turn become osteoblasts together forming bone remodeling units that control resorption and formation. In this theory, bone formation must be preceded by bone resorption. These two processes are necessarily balanced, though how they are linked has been obscure. Imbalanced cellular differentiation changes bone mass and/or calcium balance. Hormones and ions act by shifting the cellular balance. Calcitonin is thought to stimulate transformation of osteoclasts to osteoblasts so that bone resorption is inhibited. But bone formation is not increased because calcitonin also blocks activation of new osteoprogenitor cells. In this schema, corticoids cause osteoporosis: (1) by blocking the transformation of osteoclasts to osteoblasts, (2) by inhibiting the synthesis of bone matrix and, (3) in the presence of the parathyroids, activating osteoprogenitor cells to form osteoclasts. The authors believe that corticoid osteoporosis is mediated by secondary hyperparathyroidism, a mechanism originally suggested by Laron in 1957 and subsequently supported by Eliel (1965), Gordan (1966), Jee (1972) and recently shown directly by Williams (1973). However, in my experience, corticoids first increase accretion and lower the serum calcium level which probably accounts for secondary hyperparathyroidism.

In postmenopausal osteoporosis, insufficient transformation of osteoclasts to osteoblasts is postulated to explain the decline in active bone formation. This concept is in harmony with the classic theory of Fuller Albright and with the kinetic data of Eisenberg and Gordan (1961), Hioco et al (1964) and Lafferty et al (1964), but not with Heaney, Fraser and many others whose data have been interpreted to show normal accretion and increased osteolysis. Rasmussen and Bordier imply from their version of the Albright theory that calcitonin, fluoride and phosphonate cannot be effective therapeutic agents for treating postmenopausal osteoporosis. Similarly, calcitonin can be beneficial in Paget's disease (viewed here as neoplasia of bone remodeling units) only if it does not induce secondary hyperparathyroidism, which, unfortunately, it does.

The authors' theories are stimulating, appear to be well documented and provide a cellular basis for many well-established clinical phenomena. It is refreshing that the focus has been placed on cells in these days when classical bone pathology can hardly be said to flourish. The theory adapts well to many still unexplained and apparently paradoxical phenomena, that is, the beneficial value of corticoids and of cytotoxic chemotherapeutic agents in many hypercalcemias. The publishers have done an outstanding job. The price has been kept reasonable, in large part by reproducing 51 color microphotos in a single transparency which hopefully will not be separated from the volume. Most physicians will find the book highly specialized and theoretical, certainly too much so for most medical students. It is not a textbook of practical diagnosis and treatment. It should prove provocative and useful for workers in the rapidly burgeoning field of investigation on bones, parathormones and vitamin D-derived hormones.

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